Dissertation

On

PROSPECTIVE STUDY ON MANAGEMENT OF RECURRENT GIANT CELL TUMORS AND AGGRESSIVE GIANT CELL TUMORS WITH PATHOLOGICAL FRACTURE

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CERTIFICATE

Certified that the dissertation on **"PROSPECTIVE STUDY ON MANAGEMENT OF RECURRENT GIANT CELL TUMORS AND AGGRESSIVE GIANT CELL TUMORS WITH PATHOLOGICAL FRACTURE"** is a bonafide work done by **Dr.T.M. MANOHAR,** Postgraduate, Department of Orthopaedic Surgery, Kilpauk Medical College & Hospital, Chennai - 10 under my guidance and supervision in partial fulfillment of the regulations of The Tamilnadu **Dr.M.G.R. Medical University** for the award of **M.S.Degree Branch II (Orthopaedic Surgery)** during the academic period of June - 2003 - September 2006.

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CONTENTS

INTRODUCTION

Giant cell tumor is a primary bone tumor. It is benign but locally aggressive neoplasm with a tendency for local recurrence.

The incidence of giant cell tumor in the Western World is relatively low, constituting 5 per cent of all skeletal tumors, however, in the Orient it may account for 20 per cent of all primary skeletal neoplasms. It is unknown whether genetic differences exist in different races that may account of the high incidence and different behaviour of this tumor among the South East Asian population.[61]

In the pre roentgen era, most of these tumors were treated by radical amputation. With the invention of X-ray less radical surgery was proposed and practiced.

The optimum treatment of giant cell tumor of bone is a matter of controversy. With the advent of variety of adjuvant and reconstruction techniques the recurrence rate has decreased remarkably. But there are no absolute clinical, radiological, or histological parameters that accurately predict the tendency of any single lesion to recur or metastasize.[35]

Most patients incurring a giant cell tumor of bone are young and active with normal life expectancy. The aim of treatment is to remove the tumor completely and to preserve the joint. These aims have not changed, but the methods of treatment have changed with time.

As might be expected, when feasible, curettage with preservation of the joint is to be preferred over an en bloc resection, which is associated with a higher rate of complications and less satisfactory functional results.

Local recurrence is a well documented problem. It is more common after simple curettage. 25% of the recurrences were within six months and 97% within two years[49].

AIM OF THE STUDY

This study is aimed at analysing the treatment of the recurrent GCT and aggressive GCT with pathological fracture by adequate curettage, using adjuvants like H_2O_2, liquidnitrogen, followed by filling the curetted cavity with bone grafts, bone substitutes & bone cement, thereby preventing the recurrence, and to provide structural stability in aggressive GCT with Pathological fracture.

REVIEW OF LITERATURE

HISTORY[53]

In ancient times our forebears knew only of malignant tumors. Small benign tumors were largely unknown until x-rays revealed them at the turn of the 20th century.

Sir Astley Cooper and Benjamin Travels described giant cell tumor of bone in 1818, emphasizing its essentially benign nature.

Herman Lebert (1845) identified the ubiquitous giant cells with the advent of microscope and separated giant cell tumors from metastasis lesions to bone and other solid tumors of bone that were then generally considered to be osteosarcoma.

Sir James Paget called it as a brown or myeloid tumor and gave it classic description in 1853.

Aguste Nelation (1860) pointed out local aggressiveness of giant cell tumor in a monograph. He named this entity as tumor of myeloplaxes, myeloplaxes being osteoclastic giant cells according to the then prevalent terminology.

Rudolf Virchow (1963 - 1867) described that these tumors may not only recur but also eventually turn into a fully malignant tumor.

Morris (1876) rejected a giant cell tumor from the wrist.

Joseph Blood good (1912) named it as Benign giant cell tumor and justified

the attempt of curetting to preserve function and he was the first to recommend phenol treatment of the defect.[59]

Stewart (1822) introduced the term osteoclastoma.

Henry L.Jaffe (1896- 1976) in 1940 proposed a histological grading system.[54]

Lawson (1952) reported one case of giant cell tumor of the distal radius treated with nonvascularized fibular autograft.[26]

Vidal et al. (1969) described the technique of Intralesional curettage followed by packing of the defect with PMMA.[5,6]

Person and Waders described the concept of extending the effective margins of a curettage beyond its geographic borders with the use of PMMA during mid 1970's.[30]

Marcove *et al.* (1970) pioneered the development of cryotherapy in the treatment of giant cell tumors of bone and described the effectiveness of the direct pour method in freezing the walls of the curetted cavity.[30]

Mario Champanacci proposed a radiographic grading system in 1977.[7]

William F. Enneking (1980) produced a satisfactory staging system for musculoskeletal tumors, which was based on clinical, radiological and pathological criteria and defined the extent of the surgical procedure to be performed to remove the tumor.[16]

Wilkins *et al.*, (1987) reviewed the data of heat effects of PMMA and evaluated necrosis in dog model and strongly recommended not to rely upon PMMA for tumor control and emphasized the need for thorough curettage with a mechanical burr.[30]

Johnston reported the use of H_2O_2 as a local chemical adjuvant for giant cell tumors in 1987.[37]

Schiller et al. (1989) used phenol as a chemical adjuvant and reported reduced recurrence rate as compared with surgical resection alone.

William F. Enneking (1991) devised a system for the functional evaluation of reconstructive procedures after surgical treatment of tumors of musculoskeletal system.[15]

NATURE AND GENESIS

Giant cell tumor are distinctive neoplasms because they are characterized by a profusion of multinuclear giant cells scattered throughout a stroma of mononuclear cells. The neoplastic elements are the stromal cells, not the giant cells. Giant cells do not persist in cell culture. Neither mitoses nor cellular atypia were seen in the culture.[47,58]

Three aspects of these neoplasms are of particular interest.[47]

1. Their cell of origin

2. Their differentiation from other giant cell bearing lesions.

3. Their biologic behaivour.

Studies have used *in vitro* cell culture techniques to investigate the individual cell types present in human giant cell tumors of bone. The goals has been to identify and characterize these cells, and to determine their relationship to cells of hematopoietic and connective tissue origin.[17]

Three major cell types have been identified based on cell morphology and growth characteristics, presence of specific cell surface antigens, presence of receptors for hormones and cell products released into the culture medium.[17]

The cell types are:

1. Mononuclear cells of monocyte - macrophage origin

2. Mononuclear cells of connective tissue origin

3. Multinucleated giant cells

The giant cells fulfill all the essential criteria for defining osteoclasts: They possess abundant calcitonin receptors, which respond to calcitonin with a rise in cyclic adenosine monophosphate, and are capable of forming resorption pits on bone slices in a manner identical to that of osteoclasts. The giant cells of giant cell tumors of bone are also positive for tartrate resistant acid phosphatase and have an antigenic phenotype identical to that of osteoclasts. So it can be speculated that the neoplastic stromal cells produce some factor, perhaps under the control of circulating PTH, which attracts osteoclasts into the tumor or promotes their differentiation.[3]

Differentiation of these neoplasms from other giant cell bearing lesion is as important as it is difficult. Giant cells are identified as a histologic component of the body's reaction to stimulus, provided by a foreign object such as crystalline material (monosodium urate), infections (mycobacterium and fungi), abnormal levels of hormone (PTH) and neoplasms; of which giant cell tumor is but one, where the specific stimulus that provokes the proliferation and accumulation of such cells in giant cell tumor is not known.

Age group of the patient, location in the bone, radiological features, gross

and microscopic pathological features are used to differentiate giant cell tumors from giant cell rich lesion and neoplasms such as aneurysmal bone cyst, non ossifying fibroma, brown tumor etc.[46]

Prediction of the biologic behaviour of giant cell tumors is a challenging feature. The natural history of giant cell tumor is one of continued growth and local aggressiveness, with extension beyond the cortex into the of tissues. Pulmonary metastases may occur and occasionally may spontaneously regress or can cause death from pulmonary failure.[58]

CLINICAL FEATURES

About 70 to 80 per cent of patients with giant cell tumors are between 20 to 40 years old, but the tumour is reported in patients from 5 to 73 years of age.[28] Giant cell tumors occurs slightly more often in females than in males. Eighty percent of giant cell tumors occurs in the long bones and 75% of these develop around the knee joint.

Presentations of Giant Cell Tumor

1. Typical giant cell tumor
2. Metastasizing benign giant cell tumor
3. Primary malignant giant cell tumor
4. Secondary malignant giant cell tumor.

Typical Giant Cell Tumor

Giant cell tumor is typically monostotic with predilection for the ends of long bones. The most common sites of involvement are the distal femur, proximal tibia and the distal radius, and the skeletal distribution of giant cell tumors of bone is shown in Fig.1. A rare polyostotic form of giant cell tumor exist (nearly 30 such cases were reported). The multicentric lesions may appear simultaneously or over intervals as short as 4 months to as long and 16 years. The histology and recurrence rate is similar to that of monostotic giant cell tumor.[58] Primary mutifocal giant cells tumor of bone, because of its rarity, should be a diagnosis of exclusion.[34,42]

Giant cell tumor have been noted to occur in association with paget's disease of bone. Most of the cases of giant cell tumor associated with paget's disease have bene observed in the head facial bones, spine and the pelvis.[39]

Local recurrence is a well documented problem. Recurrence can be in the bone or in the soft tissue. Rate of local recurrence depends on the site (distal radius greater than proximal tibia or distal femur) and adequacy of treatment.[58] Higher rates of recurrence have been noted in tumors of the distal end of radius, tumours with pathological fracture and Stage - III tumors according to the classification of campanacci.[38]

Goldenberg in his large series stated that 25 per cent of the recurrences were within 6 months and 97 per cent with in two years.[18]

The longest interval for recurrence that has bene described was 30 years after curettage and bonegrafting. The relative frequency of late recurrence is 1%.[49]

Metastazing Benign Giant Cell Tumor

Metastasis of the benign giant cell tumor is rare and most metastases are to the lungs; Metastases to other sites including brain, kidney, adrenal, gastro intestinal tract, other bones, skin, regional lymph nodes, the scalp, and the pelvis are extremely rare.[26] Histologically the metastases are indistinguishable from the primary tumor. Microvascular trauma resulting in tumor embolization at the time of curettage can be implicated in most patients. Although it cannot account for metastasis occurring before an operation other biological factors, including immune surveillance and intrinsic biological characterics of the tumor must be operative.[4,27]

The incidence of lung metastases to histologically proven GCT ranges from

1 per cent to 9 per cent. The mean interval between primary diagnosis and the onset of lung metastases was 4.0 years.[51]

Patients who have been managed for benign giant cell tumor of bone should be followed at frequent intervals (every 3 to 6 months) with radiographs of the chest in conjunction with monitoring for local recurrence.[27]

Patient age or gender is not a risk factor for metastases and also the site of original tumor did not influence the rate of metastasis.[51]

It is estimated that there is a six fold increase in the risk of developing lung metastases after a local recurrence.[51]

Clinical follow up of reported cases supports good prognosis and long term survival[13.]

Inoue *et al.* reported a case in which 25 nodules of lung metastases were excised from the Lt lung and 33 nodules from the right lung. All of these metastases were proved histologically to be benign GCT of bone and the patient was well without evidence of recurrence disease with an almost normal lung function. So unnecessary over treatment of lung metastases should be avoided and therapy should be limited to surgical eradication.[13]

Primary Malignant Giant Cell Tumor

Primary malignant giant cell tumor exists when a frankly sarcomatous lesion is contiguous with a typical histologically benign giant cell tumor.[8]

Secondary Malignant Giant Cell Tumor

Secondary malignant giant cell tumor results when a sarcoma develops at the site of a previously treated giant cell tumor; most of these are due to irradiation of the primary tumor.[20,33]

Dahline *et al.* reported that sarcomatous transformation occurred in 19% of patients treated with irradiation, as compared to only 3% of patients treated with other modalities.[12]

Data on treatment with orthovoltage radiation along (200 to 250 kilovolts peak) have indicated rates of malignant transformation of as high as 25 per cent.[2]

Pooled data from more recent studies on the result of treatment with a singed course of mega voltage radiation (approximately forty to seventy gray) have indicated a far lower rate of malignant transformation - less than 3 per cent.[2]

The most likely explanation for this difference between ortho voltage and mega voltage radiation is attributable to the much higher absorbed dose in bone than is indicated by the nominal prescribed dose. This phenomenon results from the physics of absorption of low energy photons, particularly the photoelectric

mechanism whereby energy absorption is directly proportional to the third power (Z^3) of the atomic number of the tissue. Hence tissues with high Z components, such as the calcium of bones, absorbs much more energy per gram of irradiated tissue than does muscles. In contrast, with radiation in the mega voltage range, the Compton effect dominates and energy absorption is independent of Z. Hence, the radiation absorption in muscle, tumor and bone does not vary as it does with the lower energy.[2]

The average latency of secondary malignant giant cell tumor is 13 years, ranging from 4 yrs to 39 yrs; and 75% are fibrosarcomas and 25% are osteosarcomas. Five year survival is 30 per cent; death mostly use to pulmonary metastasis.[48]

Signs and Symptoms

Predominant symptoms are pain and swelling of variable severity. Patients may present with decreased joint range of motion or pathological fracture.[8,10]

On physical examination, a tender hard mass in typically found. The skin over the swelling may be warm. There may be joint effusion and disuse muscle atrophy. Egg shell crackling may be present but it should not be elicited.[10]

Giant cell tumors of the spine (2% to 5%) typically involves only one vertebra and have a predilection for vertebral body, kyphosis secondary to body collapse may be evident on initial presentation; extension of the tumor into the epidural space may produce radicular symptoms and paraplegia.[12]

Giant cell tumors of sacrum (10%) are eccentric and attain large size, but rarely produce bowel or bladder dysfunction.[12] In pelvis, ilium is the most common site affected.[50] GCT in children is almost always metaphyseal.

INVESTIGATION AND DIAGNOSIS

Jaffe first emphasized and Evarts reaffirmed, the triple approach by the surgeon, pathologist and radiologist.[54]

It is imperative that the radiologist and pathologist be informed fully of all details that is available in order to maximize their contributions.

Plain Radiograph

The conventional GCT appears as a lytic lesion and is eccentrically located in epoiphyseo metaphyseal area. It is translucent, lacks stapling or calcification. They cross the epiphyseal scar and extend into the metaphysis. Periosteal reaction is absent. The radiographic hall mark of this lesion is that it abuts the subchondral bone plate of the adjacent joint. There may be progressive thinning and bulging of the cortex.[46] Perforation of the cortex is found in approximately 25% of patients and pathological fracture in 5 to 10% of patients. [8,46]

Plain radiograph is also useful in the early diagnosis of recurrence.[45] Bone recurrence usually is evident as an expanding lucency on roentgenogram.[8]

Radionuclide Scintigraphy

Giant cell tumors produce increased uptake of technetium 99mm radiopharmaceutical. It is helpful in evaluating the rare patient with multiple lesions.[12,46]

Angiography

Majority of giant cell tumors are hypervascular but approximately 10 per cent may be completely avascular. Vascularity per se does not correlated with the clinical course or predict either local recurrence or metastatic potential. It is used to determine the relationship of very large tumors to major vessels.[12,46]

Computed Tomography

Superior to conventional radiograph in outlining tumor extent and cortical continuity.[12,46]

Magnetic Resonance Imaging

Giant cell tumors exhibit signals of low intensity of T1 and high intensity on T_2 weighted images relative to the bone marrow. Therefore, the intramedullary extent is best seen on T_1 weighted images, while its extra osseous portion is best appreciated on T_2 weighted images. [12,21,46]

FNAC

It is simple to perform, inexpensive and no morbidity. it is most likely to yield a diagnostic sample if the bone lesion is lytic or bad cortical breakthrough or soft tissue extension. FNAC has been especially useful in the diagnostic confirmation of deep lesions that otherwise are difficult to access. FNAC does require clinical, radiographic and pathologic correlation. [1, 14, 60]

The predictive value of positive result is 100 per cent. So further biopsy is unnecessary. A negative result is not highly predictive (71.4 per cent).

The risk of needle track contamination of fine needle aspiration biopsy has been estimated by Papanicolaou society to be between 3 and 9 per 100,000 procedures.[14,60]

Open Biopsy

Advantages of open biopsy include familiarity, provision of more histological material and ability to analyse architecture better. If the diagnosis offered by FNAC does not fit the clinical and radiographic findings, then open biopsy should be considered.[60]

Gross pathology

The giant cell tumor is solid but often soft and friable. It replaces the bone marrow, giving it a brown or reddish appearance. Focal cystic areas, which may be composed of aneurysmal bonecystlike tissue grossly and histologically, focal yellow areas representing lipid laden macrophges and focal areas of hemorrhage and on occasion, necrosis may all be seen. Production of bone is rare.[21,47,57,58]

Histology

Solid sheet of proliferating mesenchymal cells and multinucleated giant cells scattered evenly throughout the lesion. The usual light microscopic characteristics used in defining the biologic behaviour of the tumor may be misleading in giant cell tumor of bone.

Giant cell tumors histologically may vary considerably. They range, at the benign end of the spectrum, from aneurysmal bone cysts, chondroblastoma, benign fibrous histiocytoma, non ossifying fibroma, brown tumor and giant cell reparative granuloma to malignant end of malignant fibrous histiocytoma or even osteosarcoma.[21,47,57]

Unni states that ever attempt must be made to diagnose giant cell tumor when the clinical picture suggest it, even when the histologic features are somewhat atypical.[57]

DIFFERENTIAL DIAGNOSIS - GIANT CELL VARIANTS[46,54]

S. No.	Diagnosis	Age Group	Location in the Bone	Radiological Appearance	Gross pathology	Microscopic Features	
						Giant Cells	Stomal Cells
1.	Giant cell tumor	3rd and 4th decade	Epiphysis or metaphysis	Eccentric expanded radiolucent area	Flesh soft tissue	Abundant innumber, uniformly distributed. No mitosis, no cellular atypia	Plump or poly hedral cells with abundant cytoplasm and indistinct cell membrane
2.	Non Ossifying fibroma	First decade	Metaphysis	Eccentric oval defects	Flesh soft tissue	Focal distribution relatively small cells with few nudei	Slender, spindle cells with little cytoplasm worled pattern
3.	Aneurysmal bone lyst	1st and 2nd decade	Metaphysis of long bones and vertebral column	Eccentric blown out lesions. Soap bubble appearance	Cavity filled with blood	Focal around vascular channels with hemorrhage	Large vascular channels; slender to plump cells. Hemosiderin deposition
4.	Brown tumor of hyperparathy radium	Any age	Any where in bone	Subperiosteal subchondral and sub ligamentous resorption of bone	Fleshy tissue with cystic spaces	Focal around hemosiderin pigment	Fibrous stroma with slender spindle cells
5.	Simple bone	1st and 2nd	metaphysis	Trabeculation in	Cyst field with	Focal around	Cyst wall of fibrous tissue

S. No.	Diagnosis	Age Group	Location in the Bone	Radiological Appearance	Gross pathology	Microscopic Features	
						Giant Cells	Stomal Cells
	cyst	decade		radiolucent area	clear fluid	cholesterol crystals	and metaplastic bone
6.	Chondro blastoma	2nd decade	Epiphysis	Radiolucency with spotry opacities	Firm to Fleshy tissue	Few and Focal	Plump round or ovoid cells with pericellular calcification
7.	Fibrous dysplasia	1st and 2nd decade	Metaphysis	Ground glass appearance	Firm and Gritty	Few and Focal	Woven bone and whorled fibrous tissue, no osseoblasts,
8.	Giant cell reparative granuloma	2nd and 3rd decade	Maxila and mandible	Radiolucent focus	Soft fleshy tissue	Focal around haemosiderin pigment or hemorrhage	Slender or Plump spindle cells
9.	Ossifying fibroma	2nd and 3rd decade	Maxilla and mandible	Radiopaque	Firm and Gritty	Few and Focal	Lamellar bone, trabeculae in fibrous tissue, osteoblastic rimming
10.	Osteosarcoma	2nd and 3rd decade	metaphysis	Radiolucent or dense	soft, firm or hard	Focal distribution	Malignant cell with direct osteoid formation
11.	Chondromyoxoid fibroma	2nd and 3rd decade	Metaphysis	Eccentric, expanded, lytic	Soft to firm	focal distribution	Chondroid, myxoid and fibrous lobules
12.	Osteoblastoma	2nd and 3rd decade	Diaphysis of long bones and vertebral column	Radiolucent or dense	Haemorrhagic and gritty	Focal Distribution	Abundant osteoid with osteoblast

STAGING

Staging is the process of classifying a tumor, with respect to its degree of differentiation as well as its local and distant extent, based on clinical, radiographic and histological features in order to estimate the prognosis of the patients.

Grading represents an estimation of the likelihood of metastasis based on histological measures of cell differentiation and growth.[40]

HISTOLOGICAL GRADING SYSTEM

OF JAFFE *et al*[25,24]

Grade I - Completely benign

Showing moderately loose vascular stroma composed of spindle and ovoid cells with few or no mitotic figures and numerous giant cells.

Grade II - Borderline

Showing cytologically a very compact cellular stroma showing definite evidence of atypia, tend strongly towards recurrence and in some cases - eventually undergo malignant transformation.

Grade III - Frankly Sarcomatous

Showing a compact stroma in which stromal cells are universally pleomorphic and the giant cells are smaller and less numerous and generally metastasis.

Jaffe and Huvos intend to relate histological features with the clinical course and to predict the outcome. But this system has no prognostic value and no correlation with recurrence rate.[22,25,52]

CAMPANACCI – RADIOGRAPHIC GRADING SYSTEM[7]

Both primary and recurrent tumors graded radiographically, based on margins of the lesion. It is more reliable than histologic grading, predicts tumor behavior, especially recurrence.

Grade I - Intraosseous lesion

Tumor with well marginate border of mature bone and cortex intact or slightly thinned but no deformed

Grade II - Intraosseous lesion with cortical thinning

Tumor with relatively well defined margin without radiopaque rim.

Grade III - lesion extending extraosseously

Tumor with fuzzy borders, suggesting a rapid and possible permeative growth.

ENNEKING STAGING[8,16]

Enneking has proposed a staging system for giant cell tumors based on clinical, radiological and pathologic criteria.

Stage 1 - Latent

10 to 15% of patients, virtually symptomatic often discovered incidentally, occasionally may cause pathological fracture, has sclerotic rim on roentgenographic or CT evaluation is relatively inactive on bone scan and is histologically benign.

Stage 2- Active

70% of patients; symptomatic, often associated with pathologic fracture, has expanded cortex but no break through, is active on bone scan and is histologically benign.

Stage 3 - Aggressive

10% to 15% of patients, symptomatic, rapidly growing mass, has cortical perforation with accompanying soft tissue mass, activity on bone scan extends beyond the lesion seen on roentgenogram, shows intense hypervasculavity on angiogram but is histologically benign.

METHODS OF TREATMENT

Treatment of recurrent lesions is the same as for primary lesions. After biopsy shows that the tumor is still benign, repeat curettage or resection should be performed.

The method of treatment include intralesional curettage, using adjuvants like, liquid nitrogen. Hydrogen peroxide, and filling the cavity with bonegrafts, bone substitute and some cement.

The available methods of treatment

1. Intralesional excision

a. Curettage only

b. Curettage with bone grafting

c. Curettage with bone cementing

d. Extended curettage

2. En bloc resection

a. En bloc resection only

b. En bloc resection with reconstruction

c. En bloc resection and custom arthroplasty

d. En bloc resection and arthrodesis

3. Amputation

4. Radiotherapy

1. Intralesional excision

An intralesional procedure passes through the pseudocapsule of the neoplasm directly into the lesion. Macroscopic tumor remains and the entire operative field is potentially contaminated. Curettage is an intraleional procedure. High speed burr can be used after curettage to decrease the local recurrence rate.[31]

a. Curettage Only

Curettage alone is less successful because surgeons will be less vigorous when only utilizing curettage in order to diminish the chance of post operative fracture and high incidence of local recurrence will be the result (75%).[12]

b. Curettage and bone grafting

Indicated for giant cell tumors of proximal humerus, distal radius, distal tibia and small bones, because there are no effective arthroplasty salvage procedures available in these area. If the articular surface deteriorates fusion is indicated which requires bone stock restoration.

This procedure has a recurrence rate of about 40 per cent.[52]

c. Curettage and bone cementing

Packing with cement after curettage of a giant-cell tumor has been advocated for many reasons.[38]

- Provides immediate support and allows for intensive curettage even of large tumor cavities.

- The contrast between barium - impregnated cement and the bone makes radiographic detection of a local recurrence easier.[40]

Most specific radiological sign of recurrence is lysis of 5mm of more at the cement bone interface. This precedes clinical signs by a mean of 4 months. When there is an complete sclerotic margin around the cement there will not be any recurrence.[45]

- If recurrence occurs, other therapeutic options still exist.

- Possible direct toxic effect of the monomer on tumor cells.

Wilkins *et al.,* recommended not to rely upon hyperthermia of PMMA polymerization for tumor control which never exceeds 46 C while the bone marrow necrosis occurs at or above 60 C.

Subchondral PMMA is tolerated because cartilage derives its nutrition from synovial fluid.

With this procedure, the rate of local recurrence is 9 to 14 per cent.

d. Extended Curettage

Adjuvants such as phenol, liquid nitrogen and H_2O_2 can be used with any of the above mentioned intralesional procedures to decrease the local recurrence.

2. En bloc resection

A wide excision, commonly termed en bloc resection, includes the entire tumor, the reactive zone and a cuff of normal tissue. This is an intracompartmental procedure and may leave skip lesion. Usually requires sacrifice of the articular surface, so impairs the joint function. En bloc resection is effective in the prevention of recurrence, with rates ranging from 0 to 32 per cent.[11]

a. En bloc resection only

It is recommended for giant cell tumors of expendable bones such as proximal radius and fibula, distal ulna, tubular bones of hand and foot, coccyx, sacrum and pelvic bones.

b. En bloc resection with reconstruction

Commonly recommended for giant cell tumors of the distal radius multiple and rapid recurrences of GCT and displaced intra articular pathological fractures.

Method of reconstruction after en bloc resection of distal radius can be with:

1. Allogenic transplantation of the distal radius.
2. Iliac crest autograft.
3. Non-vascularized proximal fibular autograft.
4. Vascularized proximal fibular autograft.

c. En bloc resection and custom made arthroplasty

For the uncommon elderly patients with an extensive giant cell tumor, a massive endoprosthesis would be a consideration. However because of the high incidence of late loosening, such reconstruction is not preferred for younger patients.

d. En bloc resection and arthrodesis

Arthrodesis should be reserved for salvage of a failed arthroplasty. If more than 50% of the articular surface is destroyed a primary fusion is considered.[59]

Primary arthrodesis of wrist may be considered for patients who do heavy manual labour.[9,24]

Amputation

Amputation is reserved for massive recurrence, malignant transformation or infection.[31,41]

4. Radiotherapy[2,21]

Megavoltage radiation therapy administered in single course (50 to 60 gray).

Indications

1. When patient can not be operated on for medical reasons.

2. When a tumor is technically inoperable - axial skeleton & skull.

3. When an operation would result in major and unacceptable disfigurement.

ADJUVANTS - THEIR MECHANISMS OF ACTION AND COMPLICATION

1. Liquid Nitrogen - Cryosurgery[29,30,31,32,36]

Extreme cold is used to produce tissue necrosis

Temperatures between - 21 to - 60 are needed to obtain cellular necrosis, temperatures below -60 exert no further lethality.

The following mechanism underlie cellular injury at subzero temperatures.

1. Thermal shock
2. Dehydration and toxic effects of electrolyte changes
3. Formation of intra cellular ice crystals and membrane disruption - most important mechanism.
4. Denaturation of cellular proteins.
5. Micro vascular failure - most likely cause of late complications.

Function involved in the spread of freezing and subsequent necrosis are [32]

1. Density and vascularity of bone

2. Presence or absence of tourniquet

3. Size and temperature of the heat sink

4. Duration of freeze, and

5. The presence of cryoprotective molecules.

Limiting factors of cryosurgery are[32]

1. Size usually 5 inches is the maximum diameter capable of being adequately frozen and therefore necrotized.

2. When a tumor spilled into a joint, an en bloc resection would be preferable.

Marcove et al., described a direct pour technique in which liquid nitrogen is poured directly into a curetted tumor cavity instead of being introduced through the closed system.[23] This method has the advantage of increasing the contact of the coolant with the irregular was of a curetted cavities.

Rapid freeze and slow thaw cycle is recommended[30]

Rapid freeze causes intracellular ice crystals to form, whereas slow freeze causes cellular dehydration. Conversely, a slow thaw will cause intracellular crystallization and membrane disruption, whereas a rapid thaw will not. This is explained by the physics of crystallization. If there is slow warming, the numerous intracellular crystals will recrystallize into a few

large crystals that will damage the cell membrane upon fast warming, the intracellular crystals will melt before they can damage the cell.

Repeated freeze thaw cycles will also increase the extent of necrosis. this is due to increased conductivity of cold after the first freeze.

Marcove *et al.* stated that three freeze and thaw cycles produce tumor cell death upto 2 cms from the cavity margin.

Advantages

1. The rate of local recurrence is around 4 per cent
2. Preservation of adjacent joint
3. Avoidance of the need for extensive reconstruction by prosthetic replacement, allograft or arthrodesis.

Disadvantages

1. Wound problems - 5 to 10%
2. Late pathological fracture - 11 to 28%
3. Transient neuropraxia

2. PHENOL[6,12,56]

Phenol solution eliminates the remaining cells by non-specific coagulation necrosis, and DNA damage.

The reported concentration of phenol solution used for this purpose varies from 5% to 75%.

Curetted cavity should be filled with pure liquid phenol for 30 to 45 seconds. After removal of the phenol, the cavity is rinsed with 75 to 85% alcohol, the alcohol residue is then removed by vigorous saline lawage.

Advantage

1. Reduced penetration of phenol causes one to one and half a millimeters of bone injury and reduced rate of fracture.

2. High rate of cure and preservation of adjacent joint

Disadvantages

Phenol is toxic to the nervous system, the heart, the kidneys and the liver and is readily absorbed through skin, mucosa and open wounds.[6]

The use of concentrations higher than 5 per cent is hazardous and the lethal dose in 1gm.[56]

3. Hydrogen Peroxide[37,55]

Johnston first reported the use of H_2O_2 as a local chemical adjuvant for giant cell tumors.

H_2O_2 causes cell death by inhibiting lactate production in tumor cells.

Cell death occurs at concentration of 30mm H_2O_2 which is substantially lower than the 3% (880mm) H_2O_2 commonly used clinically.

In a follow up series of 38 patients treated with curettage, H_2O_2 adjuvant and bone cementing, the recurrence rate was 8%.

There have been no reported negative clinical effects of exposure to H_2O_2.

MATERIALS AND METHODS

This study was conducted between Aug 2003 to Feb 2006, of which, 10 cases of Aggressive GCT with pathological fracture and 10 cases of recurrent GCT was done. In the cases of Aggressive GCT with Pathological fracture, the bone was structurally unstable and had to be mechanically stabilized. Mere cortical breach does not qualify for this criteria.

Recurrent GCT - 10 Cases

SITE

Distal Femur	3
Proximal Tibla	3
Proximal fibula	1
Distal Radius	2
Meta carpal bone	1

SEX

Sex	**No. of Cases**
Male	6
Female	4

AGE INCIDENCE

10-20	20-30	30-40
1	7	2

STAGING ENNEKING SYSTEM

Stage – 2

Treatment Methods

Removal of bone cement, extended curettage, and bone cement - 3 cases.

Extended curettage with bone cement - 3 Cases.

Curettage & bone grafting - 2 Cases.

Amputation with adjuvant usage -1 Case.

Further Resection and Adjuvant used -1 Case.

Tourniquest used in all cases. Blood transfusion was not used in any of these cases.

Aggressive GCT with Pathological fracture

Site

Distal Femur	7
Proximial Tibia	3

Sex

Sex	**No. of Cases**
Male	3
Female	7

Age incidence

10-20	**20-30**	**40-50**
4	5	1

Staging Enneking

Stage 3

Treatment Method

Extended curettage with adjuvants H2O2/ liquid nitrogen and reconstruction with Fibular Strut graft, cancellous bone graft/ bone substitute/ bone cement.

SURGICAL TECHNIQUE

Intra lesional excision with extended curettage, adjuvant hydrogen per oxide and reconstruction with bone graft/bone substitute/bone cement.

Patient in supine position under regional or general anaesthesia, tourniquet (without exsanguination) was used during the procedure to decrease local bleeding and prevent blood from acting as a heat sink and being a barrier for the cryotherapy. Electrocautery was used on all soft tissue dissection because it potentially extends the margin of tumour kill. Violation of the joint cavity avoided to prevent the possibility of contamination of the joint cavity with tumor cells and potential injury to the cartilage after direct exposure of liquid nitrogen.

Out of 10 cases with pathological fracture, seven were in distal femur and three in proximal tibia, after exposure, a large elliptical cortical window with its axis parallel to the long axis of the bone was made to reduce the stress raising effect, on the side of maximum involvement and breach. Adequate window was made and the cavity was curettaged thoroughly. Hydrogen peroxide was used as adjuvant in 9 cases and liquid nitrogen in one case.

Before introduction of liquid nitrogen, bone perforations were identified and sealed. The surrounding skin, soft tissues and neuro vascular structures were protected. Large skin flaps were retracted to protect them from any possible spillage of the liquid nitrogen.

The direct pour (open) technique as described by Marcove et al. was used. Liquid nitrogen was poured through a stainless steel funnel into the tumor cavity, and care was taken to fill the entire cavity. The surrounding

soft tissues were irrigated with saline solution to decrease the possibility of thermal injury. Two freeze and thaw cycles were administered. In each cycle, liquid nitrogen was left in the cavity until it had evaporated completely. Spontaneous thaw was allowed to occur for 3-5 mts. After evaporation, the cavity was irrigated with saline. Reconstruction was performed with poly methyl methacrylate.

In patients where hydrogen per oxide was used as adjuvant, undiluted Hydrogen peroxide was used 3 times with a holding time of 3mts. each time. When sub-chondral bone is thinned or absent, cancellous bone harvested from iliac crest mixed along with bone substitutes (G-bone) were packed to a thickness of 3-5mm. Then fibular strut graft was placed across the fracture site longitudinally. If there was an inter-condylar fracture of distal femur, the strut was placed transversely and the cavity filled with bone cement.

In 10 cases of recurrent GCT, that we managed, three occurred in proximal tibia following curettage and bone cementing without any adjuvant, 3 occurred in distal femur following curettage and bone grafting without any adjuvant, One in proximal fibula following enbloc resection, another in metacarpal bone following enbloc resection and reconstruction with fibular graft and two in distal radius following curettage and bone grafting.

For proximal tibial recurrence the cement was removed and extended

curettage was done using hydrogen peroxide and fixed again with bone cement. For distal femur recurrence, extended curettage with adjuvant H_2O_2 was done and cavity filled with bone cement. Proximal fibula where resection was done previously, further segment of bone was resected and hydrogen peroxide was used to prevent recurrence.

In the case of second meta carpal bone recurrence, second ray amputation was done and hydrogen peroxide was used. Recurrence in distal radius was managed with extended curettage using hydrogen peroxide and bone grafting.

Fresh instruments, an additional layer of surgical drapes and new surgeon gloves were used to complete surgery after the tumor resection and adjuvant treatment. Wound closed with suction drain insitu.

Post operative management and follow-up

Routine antibiotics were administered for 5-7 days, the drain was removed after 48 hrs and wound was examined.

For the patients with pathological fracture, the limb was protected in a removable brace for six weeks with intermittent gentle passive mobilization done under strict supervision. After six weeks the brace was removed, full mobilization was started and gradual weight bearing allowed only after radiological evidence of union, which was between 10-12 weeks.

For the patients with recurrent GCT, the wound was examined on the

third day after surgery, if the skin was intact, passive and active motion of the adjacent joint was begun. Weight bearing allowed with support after 72 hrs.

Roentgenogram of the tumor site and the chest at 3 months intervals for 1 year, at 6 month interval for the following 2 year, and annually thereafter was taken to detect local recurrence and pulmonary metastases.

SCORING SYSTEM

The outcome was graded according to the scoring system of william.F.Enneking[15].

In brief the system assigns numerical values (0-5) for each six categories. Pain, function and emotional acceptance in the upper and lower extremities; supports, walking ability, and gait in the lower extremities; and hand positioning, manual dexterity and lifting ability in the upper limb (Appendix).

OBSERVATION & RESULTS

10 cases of Recurrent GCT and 10 cases of Aggressive GCT with pathological fracture were studied.

Out of 10 recurrent lesions, in three patients proximaltibia, in three patients distal femur, in two patients distal radius, in one patient proximalfibula and in one patient metacarpal bone were affected.

Out of 10 Aggressive GCT with pathological fracture in 7 patients distal femur and in three patients proximal tibia were affected.

For recurrent GCT, removal of bone cement, extended curettage with adjuvant Hydrogen peroxide and reconstruction with bone cement/ born graft/ amputation were the treatment methods employed.

For Aggressive GCT with pathological fracture, extended curettage with adjuvant H_2O_2/ liquid nitrogen and reconstruction with fibular strut graft cancellous bone graft, bone substitute and bone cement were the treatment methods employed.

The results were assessed with the scoring system proposed by Enneking.

Scoring After Surgery in Lower Extremely

Factor	No. of patients	Maximum attainable score	Total score	Rating percentage
Pain	17	85	82	96
Function	17	85	51	60
Emotional Acceptance	17	85	68	80
Support	17	85	70	82
Walking ability	17	85	75	88
Gait	17	85	70	82
Average				**81.3**

Scoring after surgery in upper extremity

Factor	No. of patients	Maximum attainable score	Total score	Rating percentage
Pain	3	15	15	100
Function	3	15	14	93
Emotional Acceptance	3	15	14	93
Hand Positioning	3	15	14	93
Manual dexterity	3	15	14	93
Lifting ability	3	15	13	86
Average				**93**

The average follow up was 2 years.

Two patients had superficial wound infection, healed well with antibiotics.

There was no neurovascular complications, malignant change, recurrence, pathological fracture and metastasis.

Radiologically there was no lysis at cement bone interface.

The average rating percentage for patients after surgery in lower extremity was 81.3

The average rating percentage for patients after surgery in upper extremity was 93.

DISCUSSION

Though historically it is believed that GCT occurs more commonly in females[21], there are other series which reported male predominance. C.R.R.M Reddy et al. reported 64% in males[44], and H.N. Sung et al. reported 56% in males.[52]

The factors that would have led to recurrence were inadequate curettage and lack of extended curettage with adjuvants.

Local recurrence is a well documented problem. It has been as frequent as 50% after simple curettage and 7% after excision with curettage, 0% after wide or radical resection. A report of a large series stated that 25% of the recurrences were within 6 months and 97%, within 2 years. Some authors have reported that all recurrences were within three years. But others have reported recurrences after six years, ten years, 12 years and thirteen years.[49]

In this study we treated 10 cases of Aggressive GCT with pathological fracture and 10 cases of Recurrent GCT.

Simple curettage or curettage with bonegrafting resulted in recurrence from 30 to 80%.[52] Various adjuvants such as electro-cautery, phenol, liquid nitrogen, hydrogen peroxide have reduced the recurrence rate to 10%.[55]

Jonston in 1987 first reported the use of hydrogen peroxide in GCT.[37]

Hydrogen peroxide is preferred over other adjuvants because it is less toxic to surrounding tissues, has the same efficacy in terms of preventing recurrence[55] and in vitro studies have demonstrated that in lesser concentration, it produces cell lysis and death.[37]

Patients presenting with pathological fracture and loss of cortical bone support of less than 50% of the cross sectional area of the bone, extended curettage with PMMA reconstruction is suggested[59]. Sung et al described a procedure of excision and curettage in such cases where the main bulk of tumor is excised, retaining the articular cartilage covered by a thin shell of bone and then remaining cavity curetted. In their 12 cases they have not had any recurrence[52].

Other forms of treatment like massive allograft construction has increased chance of infection, fracture and recurrence. In patients with GCT occurring in third decade having otherwise normal lifespan, if constrained endo-prosthesis is used, they will develop early loosening and loss of bone stalk. They will require a very complicated salvage procedure within a very short span of time.

We report 10 cases of aggressive GCT with pathological fracture with more than 50% of cortical bone involvement treated by partial excision and reconstruction with fibular strut graft and bone cement. All had good functional range of motion with an average flexion of upto 100^{o} and full weight bearing on an average of 4.5 months following surgery.

PMMA acts as a filler and provides immediate stability to the bone but it does not act as an adjuvant in reducing the rate of recurrence.[55] Recurrence occurred following reconstruction with bone cement without using adjuvant. We removed the bone cement and the cavity was currettaged following by bone cementing.

Enbloc resection resulted in similar or more recurrence than curettage, probably because of higher radiological grade or local tissue contamination. We had 2 cases of recurrent GCT out of 9 cases of enbloc resection which were done without using adjuvant. They were managed with a proximal resection and hydrogen peroxide was used as an adjuvant. Two cases of distal radius where curettage and bone grafting resulted in recurrence, we did an extended curettage using hydrogen peroxide and bone grafting.

CONCLUSION

GCTis a locally aggressive benign tumor occurring in young individuals with a normal life expectancy. If inadequately or inappropriately treated it results in considerable morbidity and recurrence.

Careful attention to soft tissue protection while using cryosurgery significantly decreased the previously published reports of high rates of infection and wound healing problem.

Hydrogen peroxide is an ideal adjuvant, which gives a comparable rate of recurrence and least local or systemic complications. Free fibular strut graft along with PMMA incorporates in the bone early and the joints can be salvaged with useful function. Enbloc resection must also be followed by adjuvant to prevent recurrence due to local tissue contamination.

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APPENDIX

ENNEKING SCORING
CRITERIA FOR EITHER EXTREMITY
PAIN

No.	**Description**	**Date**
5.	No pain	No medication
4.	Intermediate	
3.	Modest/ non disability	Non-morcotic analgesics
2.	Intermediate	
1.	Moderate/ intermittently disabling	Intermittent narcotics
0.	Sever/ Continuously disabling	continuous narcotics

Function

No.	**Description**	**Date**
5.	No restriction	No disability
4.	Intermediate	
3.	Recreational restriction	Minor disability
2.	Intermediate	
1.	Partial occupational restriction	Major disability
0.	Total occupational	Complete disability

Emotional acceptance

No.	Description	Date
5.	Enthused	World recommend to Others
4.	Intermediate	
3.	Satisfied	Would do again
2.	Intermediate	
1.	Accepts	Would repeat reluctantly
0.	Dislikes	Would not repeat

CRITERIA SPECIFIC FOR THE LOWER EXTREMITY

Supports

No.	Description	Date
5.	None	No supports
4.	Intermediate	Occasional use of brace
3.	Brace	Mostly branch
2.	Intermediate	Occasional cane/ Crutch
1.	One cane or Crutch	Mostly cane/ Crutch
0.	Two canes/ Crutches	Always Canes Crutches

Walking ability

No.	Description	Date
5.	Unlimited	Same as Preoperative
4.	Intermediate	
3.	Limited	Significantly less
2.	Intermediate	
1.	Inside only	Cannot walk outside
0.	Not independently	Can walk only with assistance or whell chair bound

Gait

No.	Description	Date
5.	Normal	No alteration
4.	Intermediate	
3.	Minor cosmetic	Cosmetic alternation only
2.	Intermediate	
1.	Major cosmetic	Minor functional deficit
0.	Major handicap	Major functional deficit

CRITERIA SPECIFIC FOR THE UPPER EXTREMITY

Hand Positioning

No.	Description	Date
5.	Unlimited	180 elevation
4.	Intermediate	
3.	to above shoulder or no prosupination	90 elevation
2.	Intermediate	
1.	Not above wrist	30 elevation
0.	None	0 elevation

Manual dexterity

No.	Description	Date
5.	No limitations	Normal dexterity and sensitibility
4.	Intermediate	
3.	Loss of fine movements	CAN NOT BUTTON TC OR MINOR LOSS OF SENSITIVITY
2.	Intermediate	
1.	Can not pinch	Major sensory loss
0.	Can not grasp	Anesthetic hand

Lifting ability

No.	Description	Date
5.	Normal load	Matches normal
4.	Intermediate	Less than less Normal
3.	Limited	Minor load
2.	Intermediate	Gravity only
1.	Helping only	Can not over come gravity
0.	Can not help	Can not move

MASTER CHART
RECURRENT GCT

S. No	Name	Age & Sex	Location of tumor	Time to recurrence	Initial Procedure	Diagnosis Confirmed by	Substitute Procedure	Adjuvant used	Type of Anaesthesia	Duration of followup	Status at followup
1.	Rambabu	26/M	Left proximal tibia	2 Months	Curettage & Bone cement	FNAC	Cement Removal Extended curettage and bone cement	H_2O_2	SA	2 Years & 4 Months	Disease free
2.	Yesiah	23/M	Left proximal tibia	2 Months	Curettage & bone cement	FNAC	Cement Removal Extended Curettage with Bone Cement	H_2O_2	SA	2 Years & 4 Months	Disease free
3	Palani	33/M	Left distal femur	3 Months	Curettage & bone grafting	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA	2 Years & 4 Months	Disease free
4	Vasantha	17/F	Right distal femur	4 Months	Curettage & bone grafting	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA	2 Years	Disease free
5	Gunasekaran	31/M	Left distal femur	2 Months	Curettage & bone grafting	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA	2 Years	Disease free
6	Dillikumar	29/M	Left distal radius	2 Months	Curettage & bone grafting	FNAC	Extended curettage with bone grafting	H_2O_2	GA	2 Years	Disease free no lung metastases
7	Parimala	27/F	Right Proximal fibula	2 Months	Resection	FNAC	Extended Curettage Resection	H_2O_2	SA	2 Years	Disease free
8	Murugan	25/M	Right distal radius	2 Months	Curettage & bone grafting	FNAC	Extended Curettage with Bone grafting	H_2O_2	GA	2 Years	Disease free no lung metastases
9	Kokila	25/F	Left Proximal tibia	2 Months	Curettage & bone cement	FNAC	Cement removal Extended Curettage with Bone Cement	H_2O_2	SA	2 Years	Disease free
10	Lalitha	23/F	Left II Metacarpal bone	2 Months	Enbloc resection with fibular strut reconstruction	FNAC	II Ray amputation with adjuvant	H_2O_2	GA	2 Years	Disease free

MASTER CHART

AGGRESSIVE GCT WITH PATHOLOGICAL FRACTURE

S. No	Name	Age & Sex	Location of tumor	Duration of Symptoms	Diagnosis Confirmed by	Procedure done	Adjuvant used	Type of Anaesthesia
1.	Kotteswara rao	30/M	Left Lateral condyle of Femur	8 Months	FNAC	Extended Curettage Fibular Strut Graft cancellous bone graft `G' bone & bone cement	H_2O_2	SA
2.	Helena	17/F	Left proximal tibia	6 Months	FNAC	Extended curettage, fibular strut graft, cancellows graft, `G' bone and Bone cement	H_2O_2	SA
3	Visalatchi	19/F	Left distal femur	8 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA
4	Parimala	27/F	Left proximal tibia	6 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA
5	Meganathan	29/M	Right Medial Condyle of Femur	6 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA
6	Raghu	18/M	Left distal end of femur	6 Months	FNAC	Extended Curettage bone cement	H_2O_2	SA
7	Logeswari	36/F	Left Lateral Condyle of femur	6 Months	FNAC	Extended Curettage bone cement	Liquid phenol	SA
8	Jayalakshmi	30/F	Medial Condyle of Femur	6 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA
9	Sangetha	17/F	Medial Condyle of Femur	6 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA
10	Devi	27/F	Right Proximal tibia	6 Months	FNAC	Extended Curettage with Bone Cement	H_2O_2	SA

PROFORMA

Name Hospital No.

Age Sex

Address

Occupation

Date of Presentation

Symptoms and Signs

1. Pain and its characters
2. Swelling
3. Limp
4. Limitation of joint movements

Site and Side involved

history of Injury

Date of follow up

Classification (Enneking)

Stage 1 - Latent

Stage 2 - Active

Stage 3 - Aggressive

Diagnosis confirmed by

1 FNAC - 2 Open Biopsy

Methods of Treatment

For recurrent Gct: removal of bone cement, extended curettage with adjuvant Hydrogen peroxide/ liquid nitrogen and reconstruction with bone graft/ bone substitute/ bone cement.

For Aggressive GCT with pathological Fracture - Extended curettage, and reconstruction with bone graft/ bone substitute/ bone cement.

Post operative protocol

1. Drain removal at 48 to 72 hours

2. Non-weight bearing mobilization after 72 hours

3. For recurrent GCT, passive and active mobilization of adjacent joint was started after 3 days. Weight bearing with support after 6 weeks.

4. For aggressive GCT with pathological fracture limb protection by removable brace with intermittent, gentle passive mobilization under strict supervision Gradual weight bearing after radiological evidence of union.

Follow up

I. Clinical (a) Subjective criteria for either limb

1. Pain
2. Function
3. Emotional acceptance

(b) Objective criteria specific for the lower extremity

1. Walking ability
2. Gait
3. Supports

II Radiological

a. Presence of lysis more than 5 mm

b. Complete sclerotic margin all around the cement

c. Union or consolidation of bone graft.

Complications

1. Infection
2. Wound complications
3. Pathological Fracture
4. Neurovascular Complications
5. Secondary osteoarthritis

6. Recurrence

7. Joint Stiffness

CASE REPORTS

CASE 1 (Mr.Rambabu)

26 year old man came with complaints of pain in left knee 2 months duration. History of similar illness 8 months back,diagnosed as GCT Left proximal tibia and treated by curettage and bone cement.

On examination surgical scar and tenderness present over the left proximal tibia. X ray showed area of lysis between bone cement and bone indicative of recurrent GCT.

FNAC suggestive of GCT

Treated by bone cement removal,extended curettage with Hydrogen peroxide and cavity was filled with bone cement.

2 year and 4 months follow up patient clinically and radiologically disease free.

CASE 2 (Visalatchi)

A 19 year old female came with complaints of pain and swelling in the left knee of 8 months duration.

On examination bony swelling and tenderness present in the distal femur. Knee movements were painful and restricted.

X ray showed eccentric osteolytic lesion in the epiphysio-metaphyseal region of the distal femur with pathological fracture.

FNAC suggestive of GCT

Treated with extended curettage with hydrogen peroxide and cavity filled with bone cement.

At 2 year and 4 months follow up patient is disease free. Available range of movement in the knee is 0 to 100 degrees.

CASE 3 (Helena)

A 17 year old female came with complaints of pain and swelling in the left knee of 6 months duration.

On examination bony swelling and tenderness present in the proximal tibia. Knee movements were painful and restricted.

X ray showed eccentric osteolytic lesion in the epiphysio-metaphyseal region of the proximal tibia with pathological fracture.

FNAC suggestive of GCT

Treated with extended curettage with hydrogen peroxide and cavity filled with fibular strut graft,cancellous iliac bone graft ,G-bone and bone cement.

At 2 year and follow up patient is disease free. Available range of movement in the knee is 0 to 110 degrees.

CASE 4 (Kotteswara Rao)

A 30 year old male came with complaints of pain and swelling in the left knee of 8 months duration.

On examination bony swelling and tenderness present in the distal femur. Knee movements were painful and restricted.

X ray showed eccentric osteolytic lesion in the epiphysio-metaphyseal region of the distal femur with pathological fracture.

FNAC suggestive of GCT

Treated with extended curettage with hydrogen peroxide and cavity filled with fibular strut graft,cancellous iliac bone graft ,G-bone and bone cement.

At 2 year follow up patient is disease free. Available range of movement in the knee is 0 to 90 degrees.

RECURRENT GCT

Case 1

AFTER 2 MONTHS OF INITIAL PROCEDURE, CURETTAGE & BONE CEMENT

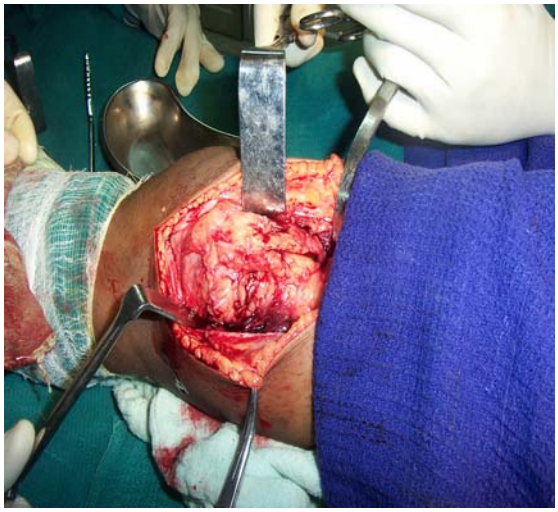
SUBSTITUTE PROCEDURE-CEMENT REMOVAL, EXTENDED CURETTAGE & BONE CEMENT

Case -2

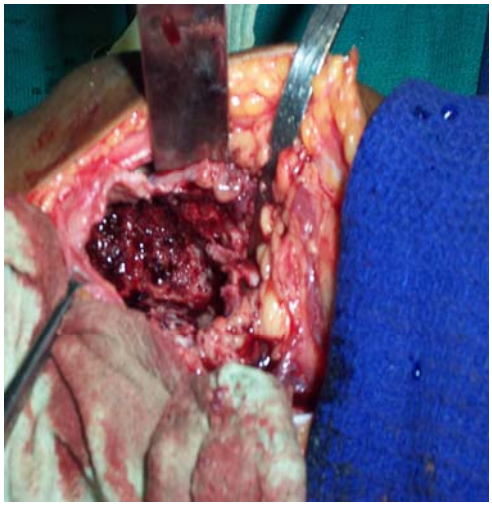
AGGRESSIVE GCT WITH PATHOLOGICAL FRACTURE - FEMUR

PRE-OP

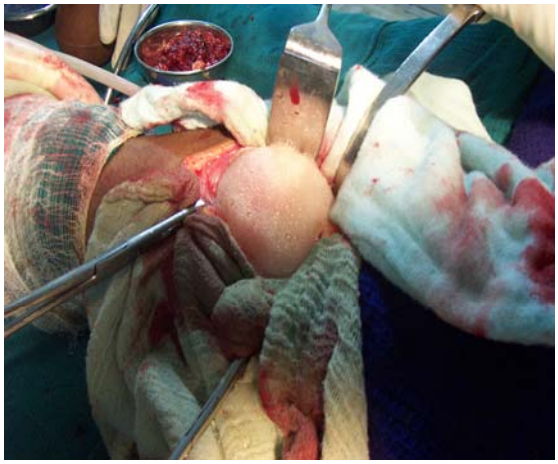
LESION AFTER EXPOSURE



THE CAVITY BEFORE CURETTAGE

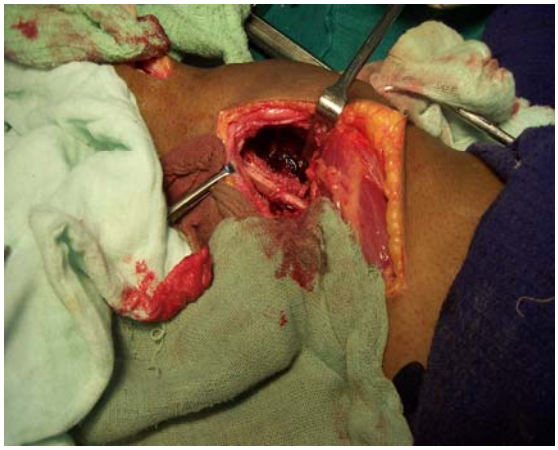


USING H_2O_2 AS ADJUVANT



CURETTED MATERIAL

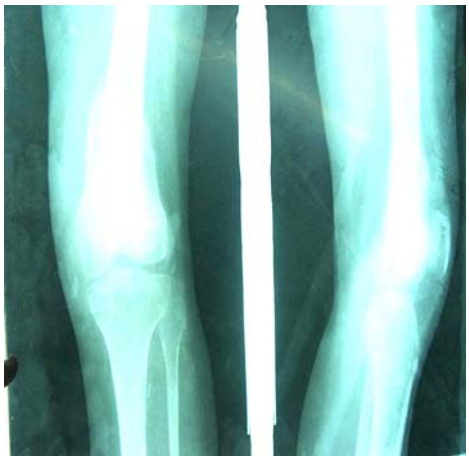
PLACING FIBULAR STRUT GRAFT



WITH BONE CEMENT

IMMEDIATE POST- OP

ONE-MONTH FOLLOW-UP



FOLLOW-UP

3 MONTHS

4 MONTHS

7 MONTHS FOLLOW-UP

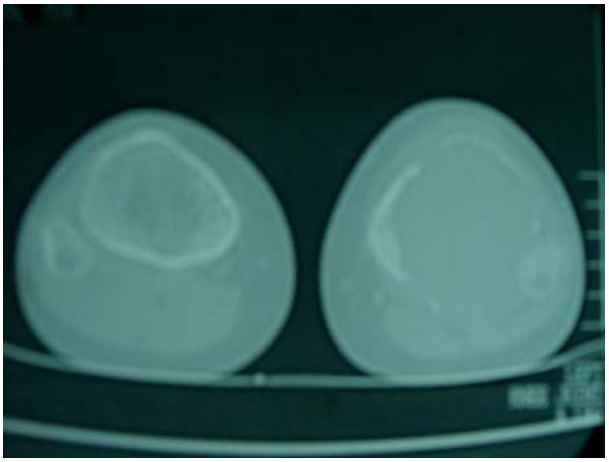
22 MONTHS FOLLOW-UP

Case -3

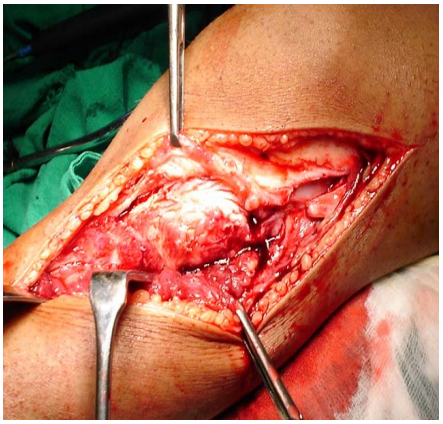
AGGRESSIVE GCT WITH PATHOLOGICAL FRACTURE – TIBIA

PRE-OP

CT SHOWING THE BREACH



LESION AFTER EXPOSURE



IMMEDIATE POST-OP

2 MONTHS FOLLOW-UP[

3 MONTHS FOLLOW-UP

6 MONTHS FOLLOW-UP

14 MONTHS FOLLOW-UP

14 MONTHS FOLLOW-UP

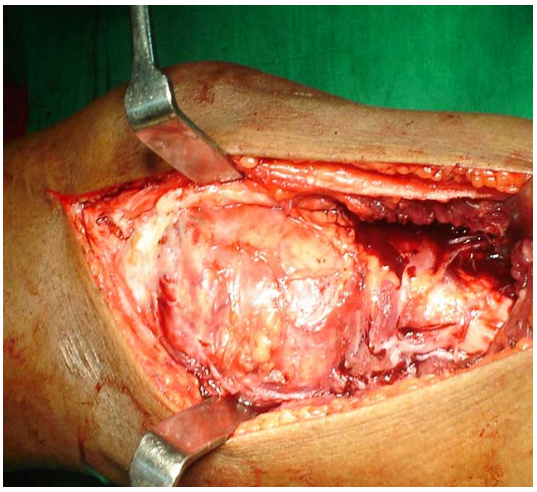
Case 4

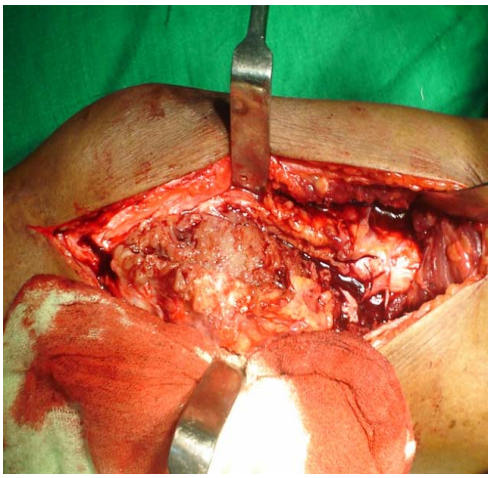
AGGRESSIVE GCT WITH PATHOLOGICAL FRACTURE – FEMUR

PRE-OP

PRE-OP

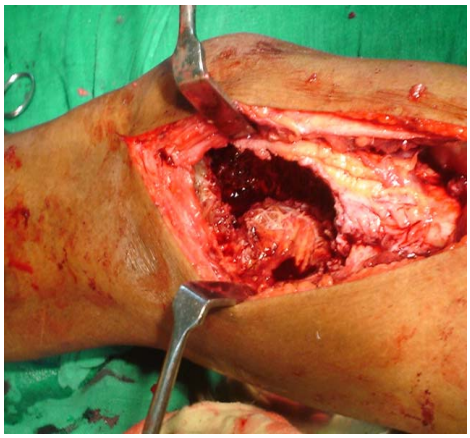
LESION AFTER EXPOSURE



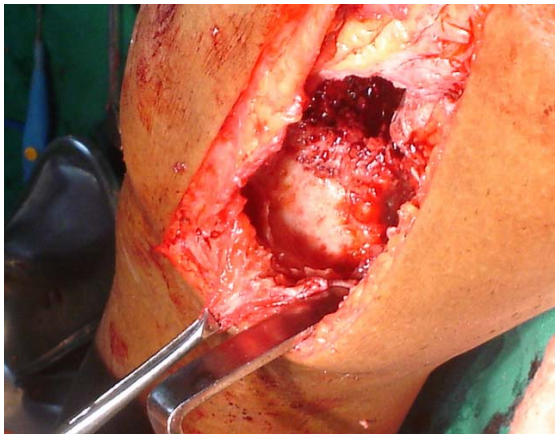


CURETTED MATERIAL

CAVITY AFTER CURETTAGE



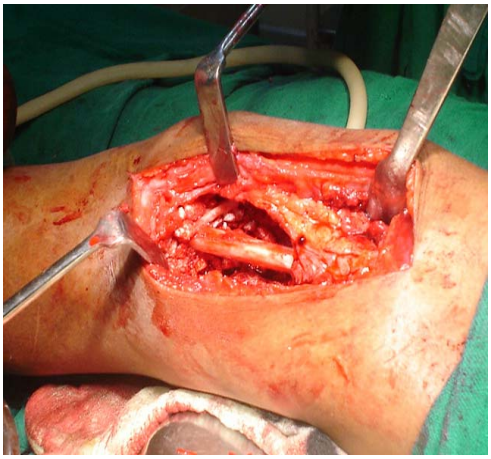
CAVITY SHOWING PATHOLOGICAL FRACTURE



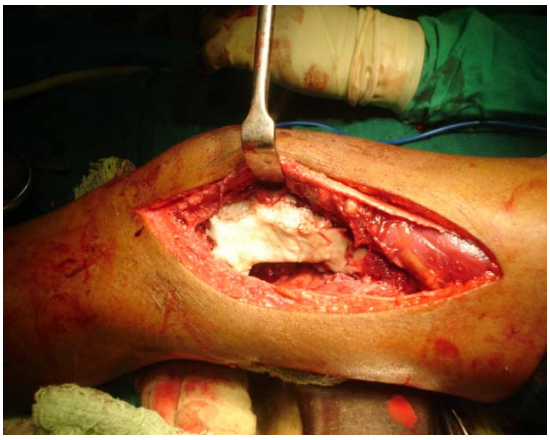
HARVESTED NON-VASCULARISED FIBULAR STRUT GRAFT

PLACEMENT OF FIBULAR STRUT GRAFT

CAVITY FILLED WITH G BONE AND CANCELLOUS BONE GRAFT



BONE STABILISED WITH BONE CEMENT



IMMEDIATE POST-OP

MICROSCOPIC PICTURE